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# INTERACTION BETWEEN SMOKING AND OCCUPATIONAL EXPOSURES

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## INTRODUCTION

There is increasing evidence of interactions between the smoking of tobacco and workplace exposures in the development of certain disease states. Most authorities believe that smoking contributes substantially to the occurrence of a spectrum of diseases including chronic respiratory disease, cardiovascular disease, and specific types of cancer, but it has not been generally recognized that occupational exposure to certain physical and chemical agents also contribute to the development of the same disease states. Some of the effects attributed to smoking may reflect an interaction between smoking and occupational exposure. This can not be quantified at the present time, but at least six different ways have been identified in which smoking may act with physical and chemical agents found in the workplace. These actions are not mutually exclusive and several may prevail for any given agent.

Six ways in which smoking may act with physical and chemical agents to produce adverse health effects are:

1. Tobacco products may serve as vectors by becoming contaminated with toxic agents found in the workplace, thus facilitating entry of the agent by inhalation, ingestion, and/or skin absorption.

2. Workplace chemicals may be transformed into more harmful agents by smoking.

3. Certain compounds in tobacco products and/or in the smoke may be the same as toxic agents found in the worklace, thus increasing exposure to the agent.

4. Smoking may contribute to an effect comparable to that resulting from exposure to toxic agents found in the workplace, thus causing an additive biological effect.

5. Smoking may act synergistically with toxic agents found in the workplace to cause a more profound effect than that resulting from the agent and smoking added together.

6. Smoking may contribute to accidents in the workplace.

Exposure to multiple physical and chemical agents in the workplace can compound these various types of actions.

ILLUSTRATIVE EXAMPLES OF DIFFERENT TYPES OF ACTIONS BETWEEN SMOKING AND OCCUPATIONAL EXPOSURES

Tobacco products may serve as vectors by becoming contaminated with toxic agents found in the workplace, thus facilitating entry of the agent by inhalation, ingestion, and/or skin absorption.

Workplace chemicals may be transformed into more harmful agents by smoking.

Investigations of outbreaks of polymer fume fever provide clear illustrations of tobacco products serving as vectors for workplace chemicals. In addition, these case studies demonstrate that workplace chemicals can be transformed into more toxic agents by tobacco smoking.

Polymer fume fever is a disease with influenza-like symptoms caused by inhalation of fumes from heated polytetrafluoroethylene (1). Typical symptoms include chest discomfort, fever, leukocytosis, headache, chills, muscular aches, and weakness. Since the symptoms are so similar to influenza, polymer fume fever is difficult to diagnose. Workers who continue to smoke may experience continuing reexposure and persisting symptomatology. Although complete recovery has usually been reported to occur within 12-48 hours after exposure is terminated, an autopsy report attributes permanent lung damage to repeated episodes of polymer fume fever (2). Pulmonary edema following exposure to heated polytetrafluoroethylene has also been reported (3,4). Polymer fume fever was first described in the literature in 1951 (5) as a result of

two workers being exposed to polytetrafluoroethylene heated to 450-500$^{0}$ C. The particular decomposition products(s) responsible for polymer fume fever have not yet been identified, but temperatures in excess of 315$^{0}$ C have been sufficient to cause symptoms. The temperature of the combustion zone of cigarettes is approximately 875$^{0}$ C (6).

Numerous outbreaks of polymer fume fever among smokers have been attributed to the decomposition of workplace polytetrafluoroethylene by lit cigarettes and inhalation of the harmful decomposition products with cigarette smoke. One report (7) describes aviation employees whose work involved contact with door seals that had been sprayed with an unspecified fluorocarbon polymer. In one case, a worker smoking during a break realized by the taste of his cigarette that it had become contaminated. Although the worker extinguished the cigarette, he experienced shivering and chills, which lasted approximately six hours, beginning a half hour after this incident. Another illustrative report (8) describes outbreaks of polymer fume fever among smoking workers whose hands were contaminated with polytetrafluoroethylene used as a mold release agent. There was no recurrence of symptoms after smoking at the plant was prohibited. An outbreak of polymer fume fever among workers using liquid fluorocarbon polymer in the production of imitation crushed velvet was likewise attributed to decomposition of fluorocarbon polymer by lit cigarettes (9). Processing temperatures at this plant were too low to pyrolyze the polymer. The seven affected workers were all cigarette smokers whereas most of the workers without symptoms were non-smokers. After work practices were changed to prohibit smoking in the work area and to require hand washing before smoking, no further symptoms at this facility were reported. [illegible] poly [illegible]

fume fever attributed to cigarette smoking have also been reported (10,11,12,13,14).

The effects of smoking cigarettes contaminated with known amounts of tetrafluoroethylene polymer have been studied with the assistance of human volunteers. (15) Nine out of ten subjects were reported to exhibit typical polymer fume fever symptoms after each had smoked just one cigarette contaminated with 0.40 mg tetrafluoroethylene polymer. Onset of symptoms ranged from 1 to 3.5 hours after smoking; recovery time averaged nine hours.

Additional research is clearly warranted to identify other workplace chemicals which are transformed into more toxic agents by tobacco smoking.

With respect to tobacco products serving as vectors, the National Institute for Occupational Safety and Health (NIOSH) has thus far identified the following agents as potential candidates for contamination of tobacco and tobacco products.

| Agent | Major Health Effects |
| --- | --- |
| Formaldehyde (16) | Respiratory irritant, dermatitis |
| Boron Trifluoride (17) | Respiratory irritant, joint disease |
| Organotin (18) | Respiratory irritant |
| Methyl Parathion (19) | Reduced erythrocyte cholinesterase activity |
| Dinitro-ortho-Creosol (20) | Kidney damage, peripheral neuritis, CNS disturbances. |
| Carbaryl (21) | Inhibition of acetylcholinesterase |
| Inorganic Fluorides (22) | Fluoride osteosclerosis |
| Inorganic Mercury (23) | CNS disturbances, kidney damage, peripheral neuritis |
| Lead (24,25) | Nervous system toxin, renal toxin, changes in hematopoietic system |

<u>Certain compounds in tobacco products and/or smoke may be the same as toxic agents found in the workplace, thus increasing exposure to the agent.</u>

Hydrogen Cyanide

Hydrogen cyanide has been found in cigarette smoke at concentrations as high as 1,600 ppm (26). In 1973 Pettegrew and Fell (27) found the plasma thiocyanate (a metabolite of cyanide) levels of smokers significantly elevated as compared to those in non-smokers. In 1973 Radojicic (28) reported a study of 43 workers in the electroplating division of an electronics firm in Nes, Yugoslavia. He found that the majority of workers exposed to cyanide complained of fatigue, headache, asthenia, tremors of the hands and feet, and pain and nausea. The urinary thiocyanate concentrations of the exposed group of workers were higher at the end of the work shift than before exposure at work. Urinary thiocyanate concentrations were significantly higher among exposed smokers than unexposed smoking controls, significantly higher among exposed non-smokers than unexposed non-smokers, and significantly higher among exposed smokers than among exposed non-smokers. These findings demonstrate that smoking and occupational exposure can each contribute to a workers' total exposure to and intake of cyanide.

Adverse effects from cyanide may occur from sublethal fatal doses. Hydrogen cyanide and cyanide salts inhibit cytochrome oxidase. Cyanide

can form complexes with heavy metal ions. Formations of these complexes in the body can rapidly cause disturbances in enzyme systems in which heavy metals act as cofactors either alone or as part of organic molecules (29,30,31). Thiocyanate itself has toxic effects, especially inhibition of uptake of inorganic iodide into the thyroid gland for incorporation into thyroxin (32). The National Institute for Occupational Safety and Health has estimated that over twenty thousand workers in seventy-five different occupational groups have potential occupational exposure to cyanide (33).

Carbon Monoxide (CO)

Cigarette smoking causes increased exposure to CO. A CO concentration of 4 percent (40,000 ppm) in cigarette smoke generates an alveolar CO concentration of 0.04 to 0.05 percent (400 to 500 ppm) which produces a carboxyhemoglobin (COHb) concentration of 3 to 10 percent (34,35,36). Goldsmith (21) estimated that the cigarette smoker is exposed to 475 ppm CO for approximately six minutes per cigarette.

In a study on the COHb levels in British steelworkers, Jones and Walters (38) found a 4.9 percent end of shift COHb saturation in non-smoking blast furnace workers compared to 1.5 percent saturation in non-smoking unexposed controls. For heavy cigarette smokers, the levels were 7.4

percent for blast furnace workers and 4.0 percent for smoking unexposed controls. The carboxyhemoglobin levels of blast furnace workers who smoked were in a critical range. Studies by Knelson (39) and Hovarth (40) have shown that levels of COHb in excess of 5 percent can cause cardiovascular alterations which are dangerous for persons with coronary heart disease.

Potential occupational exposure to carbon monoxide is great (41). Since a significant number of workers with coronary heart disease do smoke, additional occupational exposure to carbon monoxide may increase cardiovascular morbidity and mortality.

Methylene Chloride

Methylene Chloride is metabolized to CO in the body (42). Carboxyhemoglobin levels in blood increase with increasing environmental concentrations of methylene chloride as well as with increasing physical activity at the time of exposure (43,44). Maximum carboxyhemoglobin levels occur 3 to 4 hours <u>after</u> exposure is discontinued.

Mean methylene chloride concentrations of 778 ppm over a three hour exposure period produced a maximum carboxyhemoglobin level of 9.1% four hours after exposure was discontinued. Twenty hours after this exposure

the carboxyhemoglobin level remained elevated (4.4% vs. 0.8% prior to exposure) (44).

Based on these observations, prohibiting a methylene chloride worker from smoking on the job would not be sufficient to protect the worker who smokes after he leaves work from the additive exposures of CO from methylene chloride and tobacco smoke.

Other Chemical Agents

Other chemical agents found in tobacco or the combustion of tobacco products and also found in the workplace are: acetone, acrolein, aldehydes, arsenic, cadmium, formaldehyde, hydrogen sulfide, ketones, lead, methyl nitrite, nicotine, nitrogen dioxide, phenol, polycyclic compounds (26).

<u>Smoking may contribute to an effect comparable to that resulting from exposure to toxic agents found in the workplace, thus causing an additive biological effect.</u>

Coal Dust

Coal dust and cigarette smoking appear to act in an additive fashion to

produce obstructive airway disease. Although dust exposure alone plays a significant role in the development of obstructive airway disease, there is a significantly higher prevalence of obstructive airway disease in smoking miners than in non-smoking miners with the same dust exposure (45). Flow volume curve data from the use of sophisticated pulmonary function techniques suggest that non-smoking miners with dust induced chronic obstructive airway disease have decreased flow rates at higher lung volumes, whereas, smoking miners have decreased flow rates at all lung volumes (46).

Cotton Dust

Many investigators have noted that among cotton workers, cigarette smokers show increased prevalence of byssinosis when compared to non-smoking cotton workers (47,48,49,50). Cotton dust inhalation produces an acute clinical syndrome consisting of chest tightness, cough, and shortness of breath in cotton workers (51). This was formerly known as "Monday fever" since symptoms develop on the first day of work after an absence. The clinical syndrome may be accompanied by significant reduction in pulmonary function (52). The acute clinical and functional abnormalities produced by cotton dust gradually become more frequent as the disease progresses, eventually resulting in chronic obstructive airways disease (51).

In the acute phase of the illness there is a significantly greater diminution in pulmonary function in smokers than in non-smokers (48) and

the relationship of cotton dust and smoking to pulmonary dysfunction appears to be additive.

In the more severe phase of chronic obstructive airway disease, the relationship between smoking and cotton dust exposure appears to be synergistic (48).

Beta Radiation

In studies in mice when both beta radiation and cigarette tar were applied to produce carcinomas in the skin, cancers appeared six to seven months earlier than when radiation was administered alone. The shortened latent period gave an illusion of synergism which was reported in a preliminary analysis based on tumor yield at 18 months. However, at the conclusion of the experiment, the authors felt there was actually nothing more than an additive biological effect of cigarette "tar" and beta radiation (53).

Chlorine

Exposure to chlorine and cigarette smoke may cause an additive biological effect. Chester et al. (54) examined 139 men in a plant producing chlorine and sodium hydroxide by electrolysis of brine.

Fifty-five of the 139 workers had been accidentally exposed one or more times to chlorine at high concentrations and had required oxygen therapy at least once during their employment. The maximal mid-expiratory flow (MMF) values of workers with accidental chlorine exposure was compared with those of non-exposed workers for smokers and non-smokers. A significant difference in maximal mid-expiratory flow was seen when chlorine and smoking were considered as additive toxic agents. Maximal mid-expiratory flow values decrease from unexposed non-smokers (4.36) to unexposed smokers (4.13) to exposed non-smokers (4.10) and to exposed smokers (3.57).

Maximal Mid-Expiratory Flow Values of Workers with Accidental Chlorine Exposure by Smoking Category Compared to Non-exposed Workers.

| | Exposed | Non-Exposed |
|---|---|---|
| Smoker | 3.57 | 4.13 |
| Non-Smoker | 4.10 | 4.36 |

Capodaglio et al. (55) studied the diffusing capacity of the lung in workers employed in a plant for electrolytic production of chlorine and soda. He compared 52 exposed workers to 27 unexposed workers. The diffusing capacity of the lung was significantely lower in exposed smckers than in non-exposed smckers ($P \leq 0.02$), lower in exposed smckers than in exposed non-smckers, and lower in exposed smckers than in unexposed non-smokers ($P \leq 03$).

These studies show the additive effects of cigarette smcking and chlorine exposure.

Smoking may act synergestically with toxic agents found in the workplace to cause a more profound effect than that resulting from smoking and the agent added together.

ASBESTOS AND SMOKING

Asbestos provides one of the most dramatic examples of adverse health effects resulting from interaction between the smoking of tobacco products and an agent used in the workplace. Asbestos, the generic term used to describe chain-silicates, was first used in Finland to

strengthen clay pottery about 2500 B.C. (56). Modern industrial use of asbestos is relatively more recent, dating from 1880 when it was used to make heat and acid resistant fabrics (57,58). From that beginning its usefulness has grown immensely with its output having increased over one thousand-fold in the past sixty years (56).

With increasing industrial importance has come an increasing awareness of the adverse health consequences incurred by working with asbestos. Early in the twentieth century asbestosis was first reported and subsequent individual observations and epidemiological studies have well defined the association of this non-malignant respiratory disease and asbestos exposure. In 1935 Lynch and Smith reported a suspected association between asbestosis and lung cancer (59). Succeeding epidemiologic studies have given significant support to these early reports.

In 1968 a study of insulation workers by Selikoff et al. (60) defined cigarette smoking as an additional hazard to the health of workers exposed to asbestos. In a study of 370 asbestos insulation workers, Selikoff found that of 87 non-smokers, none died of bronchogenic carcinoma, while 24 out of 283 cigarette smokers died of bronchogenic carcinoma. This study suggested that asbestos workers who smoke have 8 times the lung cancer risk of all other smokers and 92 times the risk of non-smokers not exposed to asbestos. This same group of insulation workers were restudied five years later (61). At that time 41 of the

283 smokers had died of bronchogenic cancer. In a larger study involving 11,656 insulation workers in the United States and Canada, 134 deaths due to lung cancer were found among 9590 men with a history of regular cigarette smoking. Of the 2066 non-cigarette smokers followed over the same five year period, two deaths were due to lung cancer.

Over a ten year period, Berry et al. (62) studied 1300 male and 480 female asbestos factory workers in whom a smoking history was known. The male and female groups were then evaluated considering whether they had low to moderate or high asbestos exposure. The researchers found no significant excess deaths from lung cancer in either smoking or non-smoking groups at low to moderate exposures. However, a highly significant increase in lung cancer deaths was seen in the severely exposed who also smoked.

The above mentioned studies and other similar studies have shown that cigarette smoking and asbestos exposure together are associated with extremely high rates of lung cancer. But what role does each play in this process? Two general hypotheses have been proposed to answer this question (62). The additive hypothesis suggests that asbestos exposure and cigarette smoking act independently to produce lung cancer and that the excess risk seen when both are experienced together is due to the sum of their risks. The multiplicative (synergistic) hypothesis contends that each of the involved risk factors has a certain value for its risk and that the product of these two risks (asbestos exposure x

cigarette smoking) describes how they work together to bring about a certain result (lung cancer). Selikoff's data suggests a synergistic effect. However, in the study by Berry et al. (62), the male data does not fit either hypothesis while the female data easily supports the multiplicative hypothesis. A more recent study by Martischnig et al. (63) of 201 men with confirmed bronchial carcinoma was much less consistent with the multiplicative hypothesis and pointed more closely to the additive hypothesis. Regardless of whether the action is additive or synergistic, a substanial risk faces smokers who are exposed to asbestos.

Other neoplasms have been associated with exposure to asbestos but appear to be independent of smoking habits. Eighty-five to ninety percent of mesothelioma have been atributed to exposure to asbestos (64). The relationship of pleural and peritoneal mesothelioma to smoking and asbestos exposure was investigated by Hammond and Selikoff (61). Calculations from their studies reveal 0.38 deaths from pleural mesothelioma per 1000 man years of observation among asbestos exposed cigarette smokers and 0.39 for exposed non-smokers. Rates for peritoneal mesothelioma were 0.73 for smokers and 0.83 for non-smokers (65).

In 1971 Weiss (66) explored the relationship of asbestosis to cigarette smoking. He examined 100 asbestos textile workers by chest x-ray and

questionnaire. Pulmonary fibrosis was found in 40% of 75 workers who smoked and 24% of 25 non-smokers. Weiss determined that age, sex, and duration of exposure to asbestos were not responsible for the difference noted. Seventy-three of the above cigarette smokers were then questioned concerning amount and duration of smoking. The prevalence of fibrosis was 23% of 13 workers who smoked less than one pack per day and 43% of 60 who smoked one or more packs per day. Of 18 workers who smoked a pack or more per day for less than 20 years and had less than 20 years of asbestos exposure, 28% had fibrosis. Of 19 workers who smoked more than 20 years and with more than 20 years of exposure to asbestos, 74% had fibrosis. This study demonstrates that the prevalence of pulmonary fibrosis increases with increasing amount and duration of cigarette smoking and with increasing duration of exposure to asbestos. Due to the small size of the group he was working with, Weiss was unable to determine whether cigarette smoking and asbestos exposure were working in an additive or multiplicative manner. A study recently published by Weiss and Theodos indicates that type of asbestos as well as smoking habits are factors in the development of pleuropulmonary disease in asbestos workers (67).

In summary, workers exposed to tobacco smoke and asbestos experience far greater levels of lung cancer than would be expected from the contribution of either tobacco smoke or asbestos alone. However, other adverse health effects of occupational exposure to asbestos (e.g.,

mesothelioma) appear to be independent of smoking habits. Thus, smoking varies in its contribution to the development of different adverse health effects resulting from occupational exposure to a particular occupational agent.

## Exposures in the Rubber Industry

In a study of rubber workers, Lednar et al. (68) reported that smokers exposed to fumes and dust, particularly talc and carbon black, had a significantly higher risk of developing a pulmonary disability than did non-smokers. The combination of smoking and occupational exposure significantly elevated the probability of developing an early pulmonary disability. The authors reported that exposure to dust and smoking was associated with 10 to 12 times the risk of pulmonary disability retirement as a non-smoking, non-occupationally exposed rubber worker. This elevated risk was found where there were exposures to respirable particulates and/or solvents. This study suggests that smoking and occupational exposures in the rubber industry are synergistic since the authors report that a rubber worker who smoked and was exposed to talc had an excess relative risk of 3.40 whereas an excess relative risk of 1.77 would be expected if the effects of smoking and work exposure were additive. The mechanism of this interaction is not yet understood.

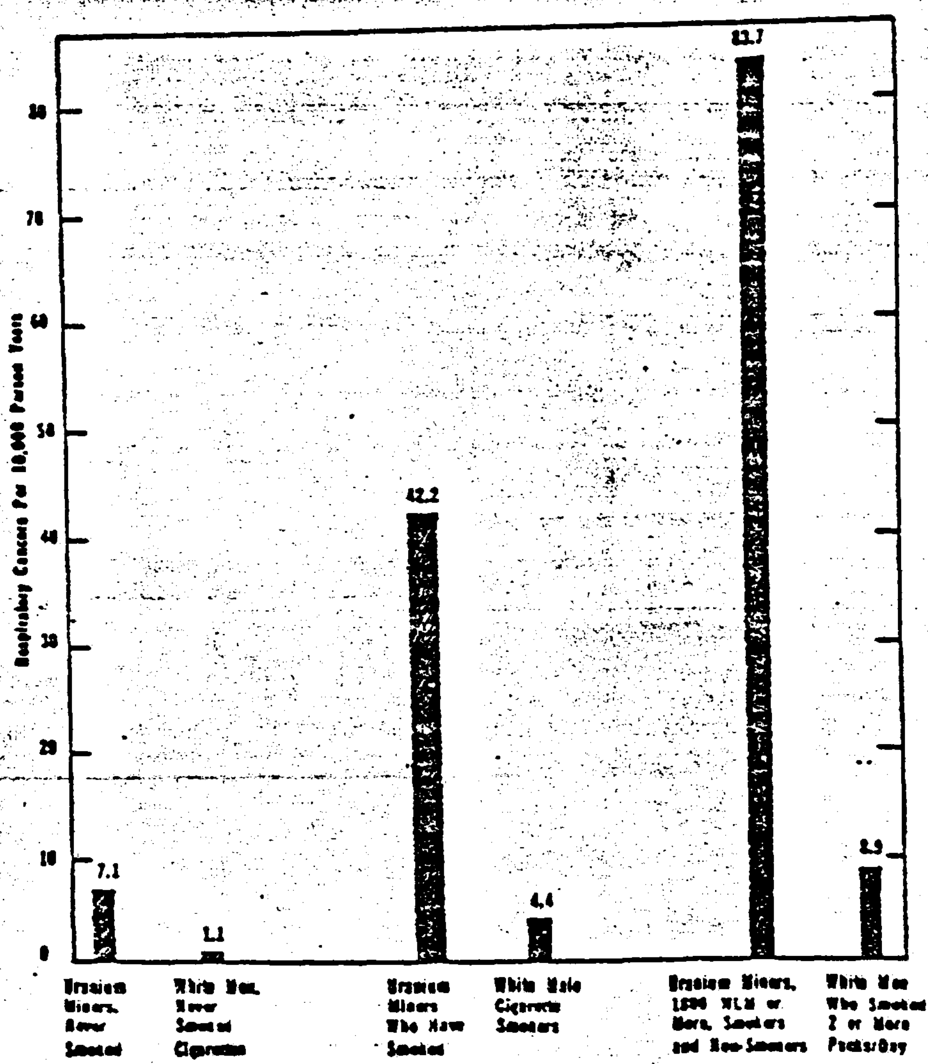
## Radon Daughters

A substantial excess of lung cancer, reduced pulmonary function, and emphysema has been reported among uranium miners (69). The excess has been attributed primarily to irradiation of the tracheobronchial epithelium by alpha particles emitted during the decay of radon and its daughter products. In a study of uranium miners, Archer et al. (70) found that respiratory cancer rates among smoking and non-smoking uranium miners were six to nine times greater than among non-miners with similar smoking habits. The lung cancer rate for nonsmoking uranium miners was 7.1 per 10,000 person years compared to 1.1 for non-miners who did not smoke. The lung cancer rate for uranium miners who smoked was 42.2 per 10,000 person years compared to 4.4 for non-miners who smoked 2 or more packs of cigarettes a day (Figure 1). There was also a definite association between the prevalence of emphysema and the cumulative amount of cigarettes smoked as well as with accumulative radiation exposure.

## Smoking may contribute to accidents in the workplace

Studies have shown that smoking contributes to accidents in the workplace. In a nine-month study of job accidents, the total accident rate was more than twice as high among smokers as among non-smokers (71). Other authors have suggested that injuries attributable to

Fig. 1 - Respiratory Cancer Rates Among Uranium Miners by Cigarette Usage and Radiation Exposure Compared with Rates Among Non-Miners*




*From: Archer V.E., Wagoner, J.K., and Lunden, F.E., Jr. "Uranium Mining and Cigarette Smoking Effects on Man". Journal of Occupational Medicine, 15(3): 204-211, March 1973.

1002600290

Editor..........IRVING R. TABERSHAW. M.
Executive Editor....DORIS L. FLOURNOY. M.

# JOURNAL OF OCCUPATIONAL MEDICIN

OWNED AND PUBLISHED BY THE AMERICAN OCCUPATIONAL MEDICAL ASSOCIATION

150 NORTH WACKER DRIVE • CHICAGO, ILLINOIS 60606 • 312/782-2166

June 14, 1978

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Dear Dr. Stein:

We are pleased to grant you permission to reproduce Figure 1 from the article "Uranium Mining and Cigarette Smoking Effects on Man" by Drs. Archer, Wagoner, and Lundin as outlined in your letter of June 8. This permission is contingent upon the authors' approval and is non-exclusive for one time use only with appropriate credit to the authors and the Journal of Occupational Medicine.

Sincerely yours,

Doris Flournoy
Executive Editor

DF:bp

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Dear Dr. Stein:

Thank you for sending me a copy of your letter to D. L. Flournoy of J.O.M. requesting permission to reproduce a figure from my article, "Uranium Mining and Cigarette Smoking Effects in Man".

Subject to approval by the editor of J.O.M., I am happy to grant you my approval.

Sincerely,

Victor E. Archer, M.D.
Medical Director

smoking were caused by loss of attention, preoccupation of the hand for smoking, irritation of the eyes, and cough (72).

Smoking can also contribute to fire and explosions in occupational settings where flammable and explosive chemical agents are used. In many of these areas smoking is prohibited. For example, smoking is not permitted in coal mines and miners are personally fined if in violation of this provision.

EXAMPLES WHERE ACTION BETWEEN SMOKING AND OCCUPATIONAL EXPOSURE HAS BEEN SUGGESTED OR ONLY HYPOTHESIZED

Cadmium

Several studies of the effects of occupational exposure to cadmium on smokers and non-smokers have been conducted (73,74,75,76,77). Pulmonary function is poorer in smokers than in non-smokers exposed to cadmium and smokers also had a higher incidence of proteinuria than did non-smokers in a cadmium exposed population in a Swedish battery factory. An additive rather than a potentiating effect seems more likely from the limited data.

Chloromethyl Ether

A group of 129 men in a chemical plant where chloromethyl ether was used were screened by 70 mm chest photofluorograms and questionnaires regarding age, smoking habits, and respiratory symptoms at intervals averaging 8.5 months for five years and follow-up for an additional five years (78). Each job classification was ranked according to degree of exposure to chloromethyl ether and an exposure index was calculated for each man by cumulating the total exposure.

Chronic cough and expectoration showed a dose response relationship to chemical exposure. Chronic cough was also related to smoking but for each smoking category, chronic cough was more common for exposed than for unexposed men.

The 10 year incidence of lung cancer was dose related to chemical exposure but not related to cigarette smoking. All cancers were small cell carcinomas, occurred in men younger than 55 and had an induction-latent period of 10 to 24 years. The 10 year mortality rate in this group of workers was 2.7 times expected and lung cancer accounted for the excess number of deaths.

Bronchogenic carcinomas linked to cigarette smoking are most often squamous cell in type with long induction-latent periods and tend to occur after the age of 60. The cancers which occur in workers exposed

to chloromethyl ether are small cell in type, have short induction-latent periods and tend to appear before the age of 55. The absence of a relationship between cigarette smoking and lung cancer in this study may be due to the competing effect of chloromethyl ether which results in lung cancer in exposed workers before the long-term carcinogenic effect of cigarette smoking could be demonstrated. However, cough related to cigarette smoking appears earlier in exposed workers, thus demonstrating the action of cigarette smoking with exposure to chloromethyl ether in the development of chronic cough symptoms. This case study also points up the complex issues involved in understanding the actions between smoking and occupational exposures.

<u>beta</u>-Naphthylamine and other aromatic amines

Doll et al. found an excess risk of bladder cancer in a series of studies (79,80) of men employed in coal gas production in England and Wales. Most of the gas workers were smokers. Chemical studies showed that inside the retort houses gas workers inhaled <u>beta</u>-naphthylamine and other aromatic amines (known bladder carcinogens). Since aromatic amines are also found in cigarette smoke (26), the gas workers who smoked received exposure to bladder carcinogens from two sources. This evidence is speculative but points out the need to assess the action between smoking and exposure to aromatic amines.

TRENDS IN SMOKING HABITS AND IN MORBIDITY AND MORTALITY RATES IN OCCUPATIONAL GROUPS

Surveys (81) have shown male blue-collar workers are much more likely to smoke cigarettes than white-collar workers. While only 37% of white-collar workers were reported in 1973 to be current smokers, 51% of those in blue-collar occupations then smoked. Also, more ex-smokers are found among white-collar workers than among blue-collar workers (35% and 28% respectively). Smoking among white-collar workers dropped from 48% to 37% between 1966 and 1970; during the same time period smoking among blue-collar workers dropped from 62% to 51%.

The pattern among female employees is quite different (81). There was little difference in smoking rates between white- and blue-collar female workers, 36% and 38% respectively, in the 1973 report. In addition, the smoking rates for 1966 were the same as those for 1970 in both groups of female workers. During the period studied, the increased cessation of smoking among female workers was offset by the increased initiation of smoking in the same group.

In a study by Boucot et al. (82), one hundred twenty-one new lung cancers developed among 6,136 men aged 45 and over who volunteered to report semiannually for chest x-rays and answer questionnaires about symptoms, smoking habits, etc., over a 10-year period beginning in 1951. The risk of developing lung cancer increased with increasing age, was

higher in non-whites than in whites, and bore a dose-response relationship to cigarette smoking. The highest lung cancer risk was among asbestos workers, 42.9/1000 man-years. The risk was 2.2/1000 man-years for men in occupational categories not thought to be associated with an increased risk of lung cancer. Occupational categories showing somewhat increased risk were metal workers, cooks, and automobile drivers. A higher percentage of whites, than non-whites (86.5% vs 77.4%), worked in occupations not thought to be at increased lung cancer risk.

The smoking habits in various occupational groups demonstrate ample opportunity for interaction between cigarette smoking and physical and chemical agents in the workplace. In general, those who have the highest smoking rates also have the highest risk for industrial exposures. Both the consumption of tobacco products and exposure of industrial agents increased steadily from 1920 to 1960. This is reflected in certain mortality trends. For example the United States age-adjusted mortality rate from carcinoma of the pancreas has been reported to have risen from 2.9 to 8.2 per 100,000 population from 1920 to 1965, an increment of 283%. The rise was found to be real and threefold in magnitude when adjustments were made for the aging of the population. A literature review on pancreatic cancer was conducted by Krain to determine real causes or associations for pancreatic cancer. His report indicated that only the data on industrial carcinogen exposure and cigarette smoking show both the trend and the statistical

mangitude of association to consider them as real causes or associations (83).

Since 1966 the consumption of tobacco products has decreased in blue collar workers while the number of industrial exposures have continued to increase (81,84). The increasingly higher rates of lung cancer in non-white males, independent of smoking habits, may reflect the late entry of non-whites into industrial settings and the fact that they have jobs with higher risk for occupational exposure to toxic agents.

SUMMARY AND RECOMMENDATIONS

Although precise relationships between smoking and occupational exposures cannot be quantified, the necessary data are beginning to accumulate.

From 1920 to 1966 both tobacco consumption increased as did the introduction into the workplace of chemicals with unknown biologic effects. Workers with the greatest risk of exposure to industrial agents also had the highest smoking rates. Since 1966 the consumption of tobacco products has decreased in male blue collar workers while the introduction of new chemicals into the workplace has continued to increase.

At least six different ways have been illustrated by which smoking may act with physical and chemical agents in the workplace to produce adverse health effects. These actions need not be mutually exclusive and exposure to multiple physical and chemical agents in the workplace can compound these various types of actions.

The examples of the interactions between the smoking of tobacco products and industrial exposures cited in this report indicate that a curtailment of smoking in certain occupational settings would contribute to the reduction of specific disease processes. NIOSH has therefore recommended in certain circumstances that workers exposed to particular agents refrain from smoking. However, it is important to note that in some situations (for example, radon daughters and chloromethyl ether), the contribution of occupational exposures to adverse health effects was greater than the contribution of cigarette smoking. Therefore, the curtailment of smoking in the workplace should not be done in lieu of curtailing occupational exposures to physical and chemical agents.

Recommendations

1. Studies on the health effects from smoking should take occupational exposures into consideration and vice versa. Whenever possible, studies should include data on occupationally exposed non-smoking as well as unexposed smoking controls.

2. The increasing rates of lung cancer in non-white males compared to white males should be investigated further with respect to occupational exposures and smoking habits.

3. The change in smoking habits of blue collar workers over the last decade provides an opportunity to more critically assess the contribution of smoking vs. occupational exposure to certain disease states. Prospective cohorts should be identified and followed for this purpose.

4. Workplace agents should be identified which interact with the smoking of tobacco to produce adverse health effects.

5. More studies on the modes of synergism between smoking and occupational exposures are needed.

6. The impact of the combination of smoking and workplace exposures upon reproductive disorders merits further study.

7. The impact of smoking in the workplace upon accidents merits further study.

8. The lack of information on the effect of side stream smoke in the development of occupational disease in non-smoking workers merits attention.

Sources recommendations 6, 7, + 8 ?

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